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Prepregnancy adherence to dietary recommendations for the prevention of cardiovascular disease in relation to risk of hypertensive disorders of pregnancy

Mariel Arvizu,[1] *Jennifer J Stuart,*[2,3] *Janet W Rich-Edwards,*[2,3,4] *Audrey J Gaskins,*[5] *Bernard Rosner,*[4,6] *and Jorge E Chavarro*[1,2,4]

[1]Department of Nutrition, Harvard TH Chan School of Public Health, Boston, MA, USA; [2]Department of Epidemiology, Harvard TH Chan School of Public Health, Boston, MA, USA; [3]Division of Women's Health, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA; [4]Channing Division of Network Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA; [5]Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta, GA, USA; and [6]Department of Biostatistics, Harvard TH Chan School of Public Health, Boston, MA, USA

ABSTRACT

Background: It is unclear whether adherence to diet recommendations for the prevention of cardiovascular disease (CVD) in the general population is also related to the risk of hypertensive disorders of pregnancy, including pre-eclampsia and gestational hypertension (GHTN).

Objectives: The aim was to evaluate the relation of prepregnancy adherence to the American Heart Association (AHA) diet recommendations and the Dietary Approaches to Stop Hypertension (DASH) with the risk of pre-eclampsia and GHTN.

Methods: Between 1991 and 2009, we prospectively followed 16,892 singleton pregnancies among 11,535 women who participated in the Nurses' Health Study II. Prepregnancy diet was assessed every 4 y, from which we calculated dietary pattern scores from the DASH diet (8 components) and the diet recommendations from the AHA 2020 Strategic Impact Goals (primary score: 5 components; secondary score: primary score plus 3 components). Pregnancy outcomes were self-reported, and we estimated the RR (95% CI) of pre-eclampsia and GHTN with log-binomial regression using generalized estimating equations to account for repeat pregnancies and adjusting for potential confounders.

Results: Women had a mean (SD) age of 34.4 (34.0) y at pregnancy. Pre-eclampsia was reported in 495 (2.9%) pregnancies and GHTN in 561 (3.3%) pregnancies. The RR (95% CI) of pre-eclampsia for women in the highest quintile of the DASH was 0.65 (0.48, 0.87) compared with women in the lowest score quintile. A similar inverse trend was observed for the AHA primary (0.74; 95% CI: 0.55, 1.00) and secondary (0.81; 95% CI: 0.61, 1.07) scores comparing women in the highest versus the lowest score quintile. Neither the DASH nor the AHA scores were related to GHTN.

Conclusions: Women with higher adherence to dietary recommendations for the prevention of CVD in the general population had a lower risk of pre-eclampsia—a common pregnancy complication related to higher CVD risk among women—than women with lower adherence to these recommendations. *Am J Clin Nutr* 2020;112:1429–1437.

Keywords: pre-eclampsia, dietary patterns, hypertensive disorders of pregnancy, gestational hypertension, sugar-sweetened beverages, AHA 2020 Strategic Impact Goals, DASH

Introduction

Pre-eclampsia and gestational hypertension (GHTN) are hypertensive disorders that develop in 6–8% of pregnancies (1) and account for 10–15% of maternal deaths worldwide (2). In the United States (3), pre-eclampsia is one of the leading causes of maternal death, with healthcare costs of up to $731 million during 2014 (4). Between 10% and 50% of women with GHTN also develop pre-eclampsia (5, 6). Whether pre-eclampsia and GHTN are independent diseases with shared phenotypical

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Supplemental Tables 1–5 and Supplemental Figures 1–3 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/ajcn/.

Data described in the manuscript, code book, and analytic code will be made available upon request pending application and approval.

Address correspondence to JEC (e-mail: jchavarr@hsph.harvard.edu).

Abbreviations used: AHA, American Heart Association; CVD, cardiovascular disease; DASH, Dietary Approaches to Stop Hypertension; FFQ, food-frequency questionnaire; GHTN, gestational hypertension; HDP, hypertensive disorders of pregnancy; HTN, hypertension; NHS-II, Nurses' Health Study II; PPV, positive-predictive value; SSB, sugar-sweetened beverage.

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features (elevated blood pressure) or if GHTN is an early mild stage of pre-eclampsia remains unclear (7). Additionally, women with prior history of hypertensive disorders of pregnancy (HDP) have increased risk of developing hypertension (HTN), cardiovascular disease (CVD), CVD-specific mortality, type 2 diabetes, and chronic kidney disease later in life (8–10).

Specific dietary interventions, such as the Dietary Approaches to Stop Hypertension (DASH) (11) effectively reduce HTN. Whether diet patterns aimed at preventing CVD and managing blood pressure in the general population may also prevent HDP is unknown. The 3 studies (12–14) that evaluated the relation between adherence to various dietary patterns before pregnancy and risk of HDP had inconclusive results. The remaining evidence of the relation between diet quality and risk of HDP assessed diet during pregnancy (15–20), which may not be ideal given that HDP, in particular pre-eclampsia, have their origins very early in pregnancy (21). Studies focusing on diet during pregnancy may miss the relevant period of exposure. Therefore, we evaluated the association between prepregnancy adherence to the American Heart Association (AHA) and DASH dietary patterns and the risk of developing either pre-eclampsia or GHTN, among participants from the Nurses' Health Study II (NHS-II). We hypothesized that women with greater adherence to these diet recommendations would have lower risk of HDP, including pre-eclampsia and GHTN.

Methods

Study population

The NHS-II is an ongoing prospective cohort of 116,429 women aged 25–42 y at enrollment (1989). All participants reported demographic characteristics, lifestyle factors, anthropometric measures, and medical history at baseline and updated these data biennially. Diet information was collected with a validated food-frequency questionnaire (FFQ) in 1991 and every 4 y thereafter. Lifetime pregnancy information was self-reported by 76,840 women (n = 189,735 pregnancies) in 2009. Women eligible for our study were those with diet information prior to each of their singleton pregnancies lasting >20 wk (n = 20,736 pregnancies). We then excluded pregnancies with missing year of pregnancy (n = 78), diagnosis of pre-existing chronic diseases before each pregnancy (HTN not induced by pregnancy, type 2 diabetes, CVD, or cancer; n = 1412), and incomplete diet information before each pregnancy (n = 2354) (**Supplemental Figure 1**). The final study population included 16,892 pregnancies from 11,535 women of whom 10,616 women had ≥2 pregnancies during follow-up (1991–2009). Women included in our analytic sample had similar reproductive and demographic characteristics to that of the source population (**Supplemental Table 1**). The study was approved by the institutional review boards of the Harvard TH Chan School of Public Health and Brigham and Women's Hospital, and consent was implied with questionnaire completion.

Hypertensive disorders of pregnancy

The primary outcome of our study is HDP, including pre-eclampsia and GHTN, which were self-reports of pre-eclampsia ("pre-eclampsia/toxemia") and GHTN ("pregnancy-related high blood pressure") for each pregnancy in the 2009 follow-up questionnaire. In outcome-specific analyses, women reporting both pre-eclampsia and GHTN were considered as having pre-eclampsia. Recall of self-reported complications of pregnancy is considered valid when compared with medical records (22). Furthermore, the validity of pre-eclampsia has been assessed among participants of the NHS-II (23). The positive-predictive value (PPV) was 89% when comparing self-report against medical records and defining pre-eclampsia based on the American College of Obstetricians and Gynecologists 2002 diagnostic criteria (24): evidence of new-onset hypertension (≥140 mm Hg systolic or ≥90 mm Hg diastolic) and proteinuria (≥300 mg/24-h urine, dipstick ≥1+, and albumin-creatinine ratio ≥0.3) identified after 20 wk of gestational age or provider report of pre-eclampsia diagnosis.

Lifestyle information and dietary pattern scores

Women reported race/ethnicity, height, smoking status, parity, multivitamin supplement use, and prior history of infertility, pre-eclampsia, and/or hyperlipidemia at baseline and updated every 2 y (except for race and height). Marital status was reported in 1989, 1993, and 1997. Baseline height and updated body weight were used to calculate prepregnancy BMI (kg/m^2). Self-reported weight is highly correlated with measured weight in this cohort (r = 0.97) (25). Total physical activity was ascertained using a previously validated questionnaire (26).

Diet was measured with an extensively validated FFQ (27, 28). Women reported their usual intake of 131 foods and beverages in the past year. Nutrient intake was estimated as the product between each food item and their nutrient content obtained from the USDA nutrient composition database (29) and supplemented with information from food and supplement manufacturers. We used the closest FFQ preceding each pregnancy; the 1991 FFQ was used for pregnancies ending in 1992–1995, the 1995 FFQ for pregnancies in 1996–1999, and so forth. We calculated 2 dietary pattern scores based exclusively on diet intake: *1*) the dietary recommendations from the AHA 2020 Strategic Impact Goals (30) and *2*) the DASH diet (31). The AHA score was defined using a priori cutoffs and further classified as primary and secondary score (32). The primary score endorses optimal target intakes for fruits/vegetables, fish and shellfish, sodium, sugar-sweetened beverages (SSBs), and whole grains. The secondary score consists of the primary score with additional target intakes for dietary factors considered to have a less robust association with CVD, namely nuts/legumes/seeds, processed meat, and saturated fat. We used the food pattern equivalent database from the USDA to estimate conversions from AHA score portions to servings for fruits and vegetables, whole grain, fish, nuts and legumes, and processed meat (33). For encouraged foods/nutrients (fruits and vegetables, fish, whole grain, nuts/legumes), intake at or greater the optimal target intake received 10 points and no intake received 0 points. For discouraged foods/nutrients (SSBs, processed meat, sodium), 10 points were allocated when intake was at or less than the optimal target intake and 0 points when intake was higher than that of the 80–90th percentile of intake among US adults (32). The remaining intakes were ranked between 1 and 9. The DASH score allocates 1 to 5 points for each component based on quintiles of intake within the study population. Women received 5 points if

TABLE 1 Dietary components from the dietary recommendations from the AHA 2020 Strategic Impact Goals (30) and the DASH diet (31)[1]

Dietary pattern scores	Highest score	Lowest score	Remaining intake
AHA primary score (32)	10 points	0 points	Ranked from 1 to 9 points
1. Fruits and vegetables, cups/d	≥4.5	0.0	<4.5 to >0.0
2. Whole grain, oz-equivalents/d	≥3.0	0.0	<3.0 to >0.0
3. Fish and shellfish, 100-g servings/wk	≥2.0	0.0	<2.0 to >0.0
4. SSBs, fluid oz/d	≤5.1	>16	≤16 to >5.1
5. Sodium, g/d	≤1.5	>4.5	≤4.5 to >1.5
Sum components from 1 to 5	Possible total score: 0–50		
AHA secondary score (32)			
6. Nuts, seeds, legumes, 50-g servings/d	≥1.0	0.0	<1.0 to >0.0
7. Processed meat, oz/d	≤0.5	>1.8	≤1.8 to >0.5
8. Saturated fat, %E/d	≤7.0	>15	≤15 to >7
Sum components from 1 to 8	Possible total score: 0–80		
DASH score			
Encouraged foods[2]	5 points	1 point	Ranked from 2 to 4 points
1. Fruits and juices, servings/d	≥5.0	0.0	<5.0 to >0.0
2. Vegetables, servings/d	≥2.6	0.0	<2.6 to >0.0
3. Nuts and legumes, servings/d	≥0.4	0.0	<0.4 to >0.0
4. Whole grain, servings/d	≥5.0	0.0	<5.0 to >0.0
5. Low-fat dairy, servings/d	≥5.0	0.0	<5.0 to >0.0
Discouraged foods/nutrients[3]			
6. Red and processed meats, servings/d	≤0.3	>2.1	≤2.1 to >0.3
7. SSBs, servings/d	≤0.1	>3.0	≤3.0 to >5.1
8. Sodium, servings/d	≤2.6	>3.7	≤16 to >5.1
Sum components from 1 to 8	Possible total score: 0–40		

[1]AHA, American Heart Association; DASH, Dietary Approaches to Stop Hypertension; SSB, sugar-sweetened beverage; Q5, highest quintile of intake; Q1, lowest quintile of intake; %E, percentage of total calories.
[2]Intake values represent the median intake from Q5 (highest score) and Q1 (lowest score).
[3]Intake values represent the median intake from Q1 (highest score) and Q5 (lowest score;).

they were at or above the median from the highest quintile of fruit/fruit juices, vegetables, and low-fat dairy and the lowest score if intake was at the median from the lowest quintile of intake; scoring was reversed for red/processed meats, SSBs, and sodium. The total scores for the AHA and DASH scores were then estimated by summing the points assigned to each food/nutrient (**Table 1**).

Statistical analyses

We compared differences in baseline characteristics by prepregnancy adherence to the AHA and DASH scores using Kruskal-Wallis test for continuous variables and chi-square test for categorical variables. We computed Spearman correlations between the adherence scores to assess the similarity of exposures. We estimated the RR (95% CI) of pre-eclampsia or GHTN using log-binomial regression models with generalized estimating equations using an exchangeable working correlation structure (34) to account for the correlation in outcomes between pregnancies of the same woman. In the few instances when log-binomial models did not converge we used log-Poisson models with robust variance instead (35).

To examine the relation between prepregnancy diet score adherence with the risk of HDP, pre-eclampsia, and GHTN, women were divided in quintiles according to AHA and DASH scores. Tests for linear trend were conducted by using the median values of intake in each quintile as a continuous variable (36). We also assessed the role of individual diet score components in the context of the overall diet by including continuous increases from the 10th to the 90th percentile of intake from each score component in the same model. To assess the shape of this association we modeled individual intake as increasing quintiles, and to confirm nonlinearity in the relation of each of the recommendations with pre-eclampsia we fitted intake as a restricted cubic spline (37). The presence of confounding was evaluated using prior knowledge aided by directed acyclic graphs (38). The final multivariable models included terms for the most recent covariate values preceding each pregnancy: maternal age at pregnancy [<30 (reference), 30–34, 35–39, 40–44, ≥45 y], BMI [<18.5, 18.5–24.9 (reference), 25.0–29.9, ≥30, missing], total energy intake (kilocalories/day), smoking status [never (reference), past, current, missing], physical activity [<3.0 (reference), 3.0–8.9, 9.0–17.9, 18.8–26.9, 27.0–41.9, ≥42 metabolic equivalent-hours/wk], history of infertility [yes, no (reference), missing], marital status [married, not married (reference)], race [white, other (reference)], parity [nulliparous (reference), 1, 2, ≥3, missing], multivitamin use [yes, no (reference), missing], year of pregnancy (1991, 2010), history of pre-eclampsia [yes, no (reference), missing], and gestational diabetes [yes, no (reference)].

We conducted sensitivity analyses to examine the robustness of our results. First, for the analysis of pre-eclampsia, we restricted the comparison group to normotensive pregnancies (i.e., excluding women with GHTN). Because women with prior pregnancy history might change their diet behaviors in the following pregnancy, we also restricted our analysis to women without history of pre-eclampsia at baseline (1989) and to the first pregnancy contributed to the study population. In addition,

TABLE 2 Prepregnancy characteristics among 11,535 women's first pregnancy contributed to the study across quintiles of the AHA and DASH dietary patterns scores: the Nurses' Health Study II (1991–2009)[1]

		Overall score, mean (SD), points					
		AHA primary: 30.0 (7.9)		AHA secondary: 45.0 (11.6)		DASH: 24.2 (4.9)	
	Overall	Q1	Q5	Q1	Q5	Q1	Q5
Number of women	11,535	2465	2146	2361	2194	2135	2333
Dietary pattern score, points	—	18.9 (3.6)	40.9 (2.6)	31.5 (4.9)	62.9 (4.1)	17.0 (1.9)	31.0 (2.0)
Age at pregnancy,[2] y	34.6 (3.9)	33.9 (3.9)	35.5 (3.9)[3]	33.8 (3.8)	35.5 (3.9)[3]	34.1 (3.9)	35.1 (3.8)[3]
White, *n* (%)	10,818 (93.8)	2318 (94.0)	1991 (92.8)	2225 (94.2)	2043 (93.1)	1963 (91.9)	2209 (94.7)[3]
BMI, kg/m^2	23.6 (4.3)	23.8 (4.6)	23.3 (4.0)[3]	24.2 (5.0)	22.8 (3.6)[3]	24.1 (4.9)	23.1 (3.9)[3]
Physical activity,[2] MET-h/wk	32.4 (99.2)	28.4 (106.7)	39.7 (95.1)[3]	28.5 (110.3)	41.9 (100.8)[3]	26.7 (101.4)	39.9 (89.4)[3]
Never smoker, *n* (%)	8299 (72.0)	1776 (72.1)	1517 (70.7)	1679 (71.1)	1569 (71.5)	1438 (67.4)	1711 (73.3)[3]
Married, *n* (%)	9270 (80.4)	1992 (80.8)	1705 (79.5)	1908 (80.8)	1723 (78.5)[3]	1668 (78.1)	1913 (82.0)[3]
Parity, *n* (%)							
Nulliparous	4042 (35.0)	842 (34.2)	814(37.9)[3]	770 (32.6)	871 (39.7)[3]	794 (37.2)	809 (34.7)
Parous	7123 (61.8)	1541 (62.5)	1267 (59.0)[3]	1511 (64.0)	1244 (56.7)	1277 (59.8)	1439 (61.7)
Missing	370 (3.2)	82 (3.3)	65 (3.0)[3]	80 (3.4)	79 (3.6)	64 (3.0)	85 (3.6)
Infertility diagnosis, *n* (%)	851 (7.4)	183 (7.4)	154 (7.2)	164 (7.0)	158 (7.2)	149 (7.0)	167 (7.2)
Preterm birth, *n* (%)	896 (7.8)	195 (7.9)	157 (7.3)	195 (8.3)	165 (7.5)	185 (8.7)	164 (7.0)
History of pre-eclampsia, *n* (%)	526 (4.6)	111 (4.5)	75 (3.5)	122 (5.2)	64 (2.9)[3]	106 (5.0)	85 (3.6)
History of GDM, *n* (%)	638 (5.5)	141 (5.7)	108 (5.0)	158 (6.7)	94 (4.3)[3]	156 (7.3)	111 (4.8)[3]
History of high cholesterol, *n* (%)	1394 (12.1)	319 (12.9)	227 (10.6)	320 (13.6)	237 (10.8)[3]	303 (14.2)	221 (9.5)[3]
Multivitamin use, *n* (%)	6304 (54.7)	1177 (47.8)	1295 (60.3)[3]	1099 (46.6)	1329 (60.6)[3]	916 (42.9)	1474 (63.2)[3]
Dietary intake[2]							
Total energy, kcal/d	1831 (544.7)	1686 (543.7)	2050 (509.4)[3]	1719 (548.0)	1967 (514.1)[3]	1583 (502.9)	2108 (513.9)[3]
Carbohydrate, %E/d	51.0 (7.4)	50.9 (7.8)	52.8 (6.9)[3]	47.9 (7.5)	55.5 (6.6)[3]	47.1 (7.7)	55.4 (6.7)[3]
Protein, %E/d	19.1 (3.3)	17.8 (3.2)	20.0 (3.2)[3]	18.5 (3.3)	19.3 (3.3)[3]	18.8 (3.5)	19.0 (3.1)[3]
Total fat, %E/d	30.6 (5.6)	32.0 (5.8)	28.2 (5.1)[3]	34.0 (5.3)	26.4 (4.8)[3]	34.1 (5.5)	27.0 (5.0)[3]
Caffeine, g/d	187.9 (189.3)	183.3 (190.4)	176.8 (176.1)[3]	192.2 (195.4)	170.4 (174.9)[3]	216.7 (206.8)	155.3 (165.9)[3]
Sodium, g/d	2.1 (0.4)	2.1 (0.4)	2.0 (0.3)[3]	2.1 (0.4)	2.0 (0.3)[3]	2.2 (0.4)	2.0 (0.3)[3]
Calcium, g/d	1.1 (0.4)	1.0 (0.5)	1.1 (0.4)[3]	1.0 (0.4)	1.2 (0.4)[3]	0.9 (0.4)	1.2 (0.4)[3]

[1] AHA, American Heart Association; DASH, Dietary Approaches to Stop Hypertension; GDM, gestational diabetes mellitus; MET-h, metabolic equivalent hours; Q1, lowest score quintile; Q5, highest score quintile; %E, percentage of total energy intake.

[2] Continuous variables are presented as means (SDs).

[3] $P < 0.05$ for differences across quintiles from Kruskal-Wallis test for continuous variables and chi-square tests for categorical variables.

to account for increased misclassification of exposure with greater time between diet assessment and outcome occurrence, we restricted analyses to pregnancies that occurred within 1 y of diet assessment (i.e., 1992, 1996, 2000, and 2004). Effect modification by maternal age (<30 vs ≥30 y), parity (nulliparous vs parous), smoking status (current/past smoker vs never smoker), BMI (<25 vs ≥25), and gestational age at birth [preterm (<37 wk) vs term (≥37 ws)] was evaluated using cross-product terms between these variables and linear terms of the dietary pattern scores. We also considered whether weight changes between pregnancies could address residual confounding by gestational weight gain among the subgroup of women with ≥2 study pregnancies. All data were analyzed with SAS 9.4 software (SAS Institute, Inc.).

Results

The analysis included 11,535 women who contributed a total of 16,892 pregnancies during 18 y of follow-up. There were 495 cases of pre-eclampsia (2.9%) and 561 cases of GHTN (3.3%) for a total of 1056 pregnancies complicated by HDP (6.2%). The AHA primary score ranged from 3 to 50 points, the AHA secondary score from 6 to 79 points, and the DASH score from 9 to 39 points. The AHA and DASH scores were highly correlated to each other (Spearman $r = 0.70$ for DASH vs AHA primary; $r = 0.79$ for DASH vs AHA secondary; $r = 0.86$ for AHA primary vs AHA secondary). Women who were in the highest quintile of adherence to each of the dietary pattern scores were slightly older, more physically active, had a lower BMI, and reported a higher frequency of multivitamin use (**Table 2**).

Greater adherence to the DASH score was inversely related to the risk of pre-eclampsia in unadjusted (**Supplemental Table 2**) and adjusted (**Table 3**) models. The RR (95% CI) of pre-eclampsia for women in the highest quintile of the DASH score was 0.65 (0.48, 0.87) compared with women in the lowest score quintile (P-trend = 0.01). A similar inverse trend was observed with the AHA primary (0.86; 95% CI: 0.71, 1.05) and secondary (0.81; 95% CI: 0.61, 1.07) comparing women in quintile 5 with those in quintile 1. The AHA and DASH scores were unrelated to the risk of GHTN (Table 3).

We then examined the relation of individual components of the AHA and the DASH scores (co-adjusting for the remaining components of each score) with risks of pre-eclampsia or GHTN. First, we modeled intake as a continuous linear term (**Figure 1**), and second, we relaxed the assumption of linearity by modeling intake as quintiles of intake (**Supplemental Figure 2**) and by

TABLE 3 Prepregnancy adherence to the AHA and DASH dietary pattern scores and the risk of HDP ($n = 1056$) [pre-eclampsia ($n = 495$) or GHTN ($n = 561$)] among 16,896 pregnancies in the Nurses' Health Study II (1991–2009)[1]

	Quintiles of score adherence, adjusted[2] RR (95% CI)					
	Q1	Q2	Q3	Q4	Q5	*P*-trend
AHA primary, range	3–23	24–28	29–32	33–37	38–50	
HDP	1.00	1.07 (0.90, 1.27)	0.90 (0.75, 1.09)	1.07 (0.90, 1.28)	0.86 (0.71, 1.05)	0.27
Pre-eclampsia	1.00	0.92 (0.71, 1.19)	0.75 (0.56, 0.99)	0.98 (0.76, 1.26)	0.74 (0.55, 1.00)	0.13
GHTN	1.00	1.20 (0.94, 1.53)	1.05 (0.81, 1.36)	1.14 (0.88, 1.46)	0.96 (0.73, 1.27)	0.82
AHA secondary, range	6–34	35–41	42–48	49–55	56–79	
HDP	1.00	0.89 (0.75, 1.07)	0.95 (0.80, 1.12)	0.90 (0.74, 1.08)	0.90 (0.74, 1.09)	0.32
Pre-eclampsia	1.00	0.69 (0.52, 0.90)	0.81 (0.62, 1.04)	0.74 (0.57, 0.98)	0.81 (0.61, 1.07)	0.20
GHTN	1.00	1.10 (0.85, 1.41)	1.07 (0.84, 1.38)	1.04 (0.80, 1.35)	0.98 (0.74, 1.30)	0.57
DASH, range	9–19	20–22	23–25	26–28	29–39	
HDP	1.00	1.05 (0.89, 1.26)	0.97 (0.81, 1.16)	0.89 (0.73, 1.08)	0.89 (0.73, 1.09)	0.09
Pre-eclampsia	1.00	0.83 (0.64, 1.08)	0.74 (0.57, 0.96)	0.76 (0.57, 1.00)	0.65 (0.48, 0.87)	0.01
GHTN	1.00	1.30 (1.01, 1.69)	1.23 (0.95, 1.60)	1.03 (0.78, 1.38)	1.18 (0.88, 1.57)	0.82

[1]AHA, American Heart Association; DASH, Dietary Approaches to Stop Hypertension; GHTN, gestational hypertension; HDP, hypertensive disorders of pregnancy; Q, quintile of adherence.

[2]Models were run by log-binomial generalized regression models with exchangeable correlation matrix using generalized estimating equations to account for repeat pregnancies from the same woman and adjusted for age at pregnancy, physical activity, smoking status, year of pregnancy, infertility diagnosis, marital status, race, parity, multivitamin use, history of gestational diabetes, and pre-eclampsia at baseline.

using restricted cubic splines (**Supplemental Figure 3**). Only higher intakes of SSBs and sodium, and lower intake of fruit and fruit juice, were related to a higher risk of pre-eclampsia in these analyses.

The associations of the AHA and the DASH scores with the risk of pre-eclampsia was consistent with the primary analysis when we excluded pregnancies with GHTN, women with a history of pre-eclampsia, and in analyses restricted to the first in-study pregnancy (**Supplemental Table 3**). Moreover, the inverse associations of the AHA and DASH scores with pre-eclampsia comparing quintile 5 with quintile 1 were stronger.

We also assessed whether the relation of adherence to AHA and DASH scores with pre-eclampsia differed according to baseline characteristics (**Supplemental Table 4**). We found a suggestion that the association between adherence to the AHA and DASH scores with risk of pre-eclampsia was stronger among women with a prepregnancy BMI <25, especially for the AHA secondary and DASH scores. No significant differences in the relation between adherence to dietary patterns and risk of pre-eclampsia were observed across strata of parity, smoking status, age at pregnancy, hypercholesterolemia, and gestational age at birth. Last, when we addressed the possibility of residual confounding from gestational weight gain, the relations between the AHA and DASH scores with the risk of pre-eclampsia became stronger compared with the original results (**Supplemental Table 5**).

Discussion

We found that following the DASH diet before pregnancy was related to lower risk of pre-eclampsia. Adherence to the AHA score was also inversely related to pre-eclampsia, but the association was weaker. Neither pattern was related to the risk of GHTN. The relations with pre-eclampsia were driven by intakes of SSBs, sodium, and fruits and fruit juices. These findings suggest that dietary recommendations aimed at improving HTN management in the general population may also have a role in preventing pre-eclampsia.

The lack of associations between the diet patterns examined and GHTN is worthy of consideration. For example, women with GHTN versus those with pre-eclampsia have higher plasma volumes (39), normal vascular endothelial function (40, 41), little or no endothelial cell damage (42), and less prominent signs of placental ischemia (43). As components of AHA and DASH dietary patterns have been previously related to lower levels of markers of endothelial dysfunction (44), these differences in endothelial function profile could explain the differences in association observed here. However, we cannot exclude that other mechanisms may also be at play. Also, the associations of the AHA secondary and DASH scores were stronger among leaner women than among overweight or obese women. This finding could suggest that the strong impact of excess weight on risk of pre-eclampsia may negate any benefits diet may have.

Thus, a diet that reduces systemic oxidative stress (45) and endothelial dysfunction and inflammation (46), such as DASH, may well prevent the onset of pre-eclampsia. In addition, because remodeling of the spiral arteries in the decidua begins in early pregnancy (21), prepregnancy diet might lie in the critical window of exposure compared with intake during mid- to late pregnancy. This is supported by the observation that our results were stronger when we restricted the analysis to pregnancies within 1 y of diet assessment. Intake from specific foods may also explain these relations. A diet rich in fruits and vegetables, fish, and nuts may lower markers of endothelial dysfunction (47) and minimizing consumption of SSBs may provide anti-inflammatory responses (48). Additionally, sodium intake triggers proinflammatory responses that result in left ventricular hypertrophy and microalbuminuria, good predictors of endothelial dysfunction (49).

From the studies that quantified the relation between dietary patterns with the risk of pre-eclampsia, the majority assessed

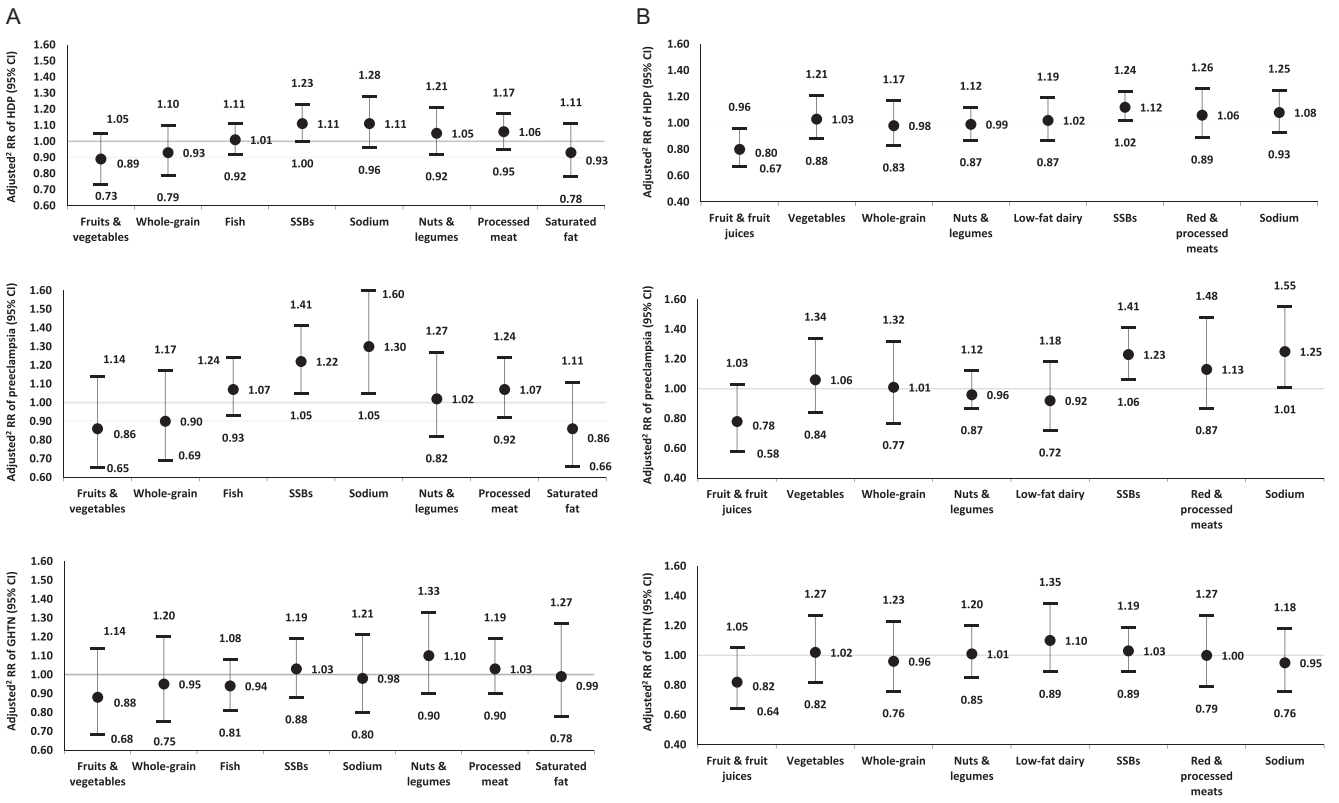

FIGURE 1 Prepregnancy increases in daily intake from the 10th to the 90th percentile of individual components from the AHA (Panel A) and DASH (Panel B) dietary pattern scores and the risk of HDP ($n = 1056$), pre-eclampsia ($n = 495$), or GHTN ($n = 561$) among 16,892 pregnancies in the Nurses' Health Study II (1991–2009). Abbreviations: AHA, American Heart Association; DASH, Dietary Approaches to Stop Hypertension; GHTN, gestational hypertension; HDP, hypertensive disorders of pregnancy; RR (95%CI), relative risk and 95% confidence interval; SSBs, sugar-sweetened beverages, E%, percentage of total calories. Models were log-binomial generalized regression models with exchangeable correlation matrix using generalized estimating equations to account for repeat pregnancies from the same woman and adjusted for age at pregnancy, physical activity, smoking status, year of pregnancy, infertility diagnosis, marital status, race, parity multivitamin use, history of gestational diabetes, and pre-eclampsia at baseline. Increases from the 10th to the 90th percentile of intake per day were as follows: sodium, 903 mg; saturated fat, 6.32%E; low-fat dairy, 2.86 servings; fruits and vegetables, 3.24 servings; SSBs, 1.28 servings; whole grain for AHA and DASH, 2.69 and 2.93 servings, respectively; fish, 0.60 servings; processed meat, 1.57 servings; red and processed meat, 1.53 servings; nuts and legumes for AHA and DASH, 0.72 and 0.88 servings, respectively; fruit and fruit juices, 3.27 servings; and vegetables, 3.54 servings.

diet during pregnancy (15–20), while only 3 women's cohorts assessed prepregnancy dietary patterns (12–14), with inconclusive results. One study among 3582 Australian women (12) found an inverse relation between pre-eclampsia and prepregnancy adherence to a Mediterranean-like dietary pattern comparing women in the highest with the lowest category of intake. However, the dietary pattern was estimated by using principal components analysis (data-derived) loading factors that explained >20% of the variation in the diet. Conversely, a dietary intervention defined by the investigators before pregnancy ($n = 297$ women) did not change the risk of developing pre-eclampsia compared with controls ($n = 285$ women) (14). Empirical estimation of diet scores may challenge the generalizability and interpretation of the results. Moreover, comparisons with our findings are not possible since the dietary pattern scores from our study were based on prespecified components and optimal target intakes from diet recommendations to prevent CVD. Last, scores from the Alternative Healthy Eating Index (aHEI-2010) had a marginal lower risk of HDP (RR: 0.84; 95% CI: 0.70, 1.02) in a recent study among American women (13). Although the associations in this study were in concordance with our findings, pregnancies <20 wk of gestational age were not excluded and HDP phenotypes were not assessed. Overall, the literature that suggests that adherence to a heart-healthy dietary pattern before pregnancy may play a role in preventing pre-eclampsia is scarce. Further evidence to confirm or refute our observations is highly recommended.

We must consider several limitations in light of our findings. Most of the pregnancies reported by our participants (85.3%) occurred before diet assessment began in 1991. Because primiparity is a risk factor for pre-eclampsia (50) and the pregnancies in our study population were mostly from parous women (70%), we had a reduced number of cases. Nevertheless, we found no evidence of effect modification by parity, suggesting that the association between diet patterns and pre-eclampsia may be comparable for primiparous and parous women. In addition, misclassification of intake is likely because diet information was only updated every 4 y. However, this misclassification is unlikely to be differential with respect to the outcome because diet was assessed prior to pregnancy, which would attenuate the associations toward the null. The occurrence of nondifferential dietary misclassification is further supported by the observation that our results were stronger when we restricted the analysis to pregnancies within 1 y of diet assessment. Additionally, analyzing the diet adherence

in quintiles rather than absolute values reduces the impact of this misclassification on the observed results, as rank is likely to be preserved over time.

Misclassification of HDP with respect to the exposure is also possible but unlikely. For example, we have no reason to believe that women with higher adherence to these dietary patterns would be more or less likely to report HDP. Moreover, the PPV (89%) of self-reported pre-eclampsia in our cohort was high and considerably higher than estimates from other validation studies (22, 51–53), which is expected in this population of healthcare workers. The underreporting of pre-eclampsia might reflect inadequate communication from physician to patient (53). Whether the complex diagnostic criteria or dynamic progression of the disease may affect self-report of pre-eclampsia is yet to be determined. Reassuringly, there is no evidence that timing of maternal recall affects the validity of self-report of pre-eclampsia, even as far out as 10 to 20 y after pregnancy (22, 51). Nevertheless, validation of self-reported GHTN is warranted and was a limitation in our study.

As is the case in any observational study, we cannot exclude the possibility of residual confounding. Despite our ability to adjust for a large number of potential confounders and the small differences in effect estimates between the unadjusted and the adjusted models, we did not have information about weight gained during pregnancy. However, we adjusted for prepregnancy BMI since self-report of periconceptional weight can be used as a reliable proxy for gestational weight gain (54). When we included additional terms for weight change between pregnancies in the multivariable model, effect estimates were unchanged for GHTN and became stronger for pre-eclampsia. We were also unable to assess early- versus late-onset pre-eclampsia; thus, we used gestational age at birth as a proxy for disease severity. Although this may be a relevant question for future research as these subtypes have differences in pathophysiology and maternal/fetal morbidity and mortality (55), it is well known that risk factors for developing early- versus late-onset pre-eclampsia are shared by both conditions (50, 55). The absence of heterogeneity within strata of gestational age at birth further supports the current literature.

Finally, the average maternal age at pregnancy was higher (34.6 y) in our study than the average maternal age for the US population at the time of the study (26.4 y) (56), which might limit the generalizability of our results. However, similar effect estimates were observed when we examined the association between diet patterns and pre-eclampsia among women <30 y versus those >30 y.

Despite these limitations, our study had many strengths, including a large sample size of women with dietary intake before pregnancy and nearly complete follow-up over the 18-y study period, the ability to distinguish between pre-eclampsia or GHTN, availability of data on multiple potential confounders, and the use of previously validated tools for diet and outcome assessment. Moreover, pegging dietary patterns to external recommendations with specific intake targets increases the generalizability of our results and allows for the possibility of direct comparison with future studies, even if dietary habits differ substantially across populations.

In conclusion, we found that greater prepregnancy adherence to the DASH diet, and to a lesser extent to the AHA diet recommendations, was associated with lower risk of pre-eclampsia. These relations appeared to be driven by lower intakes of sodium and SSBs and higher fruit/fruit juice consumption. However, neither DASH nor the AHA recommendations were related to the risk of GHTN. These findings suggest that dietary recommendations aimed at preventing heart disease and managing HTN in the general population may have a role in the prevention of pre-eclampsia. At present, there are no clinical guidelines that recommend dietary patterns for the prevention of pre-eclampsia among women at risk of pregnancy (1); therefore, diet interventions that target younger women may have the dual benefit of preventing pre-eclampsia and consequently reduce their higher risk of CVD later in life. Given the paucity of data on the relation between dietary patterns and HDP, it is imperative that further studies address the methodological issues from the majority of the existing literature.

The authors' responsibilities were as follows—MA and JEC: study conception, designed the overall research plan, and wrote the manuscript; MA: analyzed the data; JEC: had primary responsibility for final content; and all authors: critically revised the manuscript for any relevant intellectual content and read and approved the final manuscript. The authors report no conflicts of interest.

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